pranolol reduces the response to sympathetic nerve stimulation in human peripheral veins and arteries.[21,22] It is interesting to speculate that propranolol may act in RSD in part by diminishing the effects of increased sympathetic nerve activity on peripheral veins and arteries that occurs in RSD.

Herpes zoster infection can precipitate RSD in the lower extremities. This important history should be obtained in the differential diagnosis of a painful swollen extremity, especially in older adults. This case also supports the suggestion that propranolol is successful in the treatment of reflex sympathetic dystrophy and probably should be instituted early in the course of illness along with aggressive physical therapy.

Summary

Reflex sympathetic dystrophy is a syndrome characterized by painful swelling of an extremity. It is thought to result from increased sympathetic activity in the autonomic nervous system in response to an injury, but the exact cause remains unknown. A case of reflex sympathetic dystrophy of the left ankle and foot secondary to herpes zoster infection in a 72-year-old man is reported. Of particular interest was the dramatic response to propranolol therapy after conventional RSD treatment failed.

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Cardiac Arrhythmia at High Altitude

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IN PATIENTS with the sleep apnea syndrome, 24-hour electrocardiographic monitoring has shown a typical pattern of cyclic sinus arrhythmia in which the heart rate slows dramatically during apnea and increases when airway obstruction is relieved.[1,2] In this study a single subject was monitored at high altitude and this same pattern was found, suggesting that hypoxia, probably aggravated by periodic breathing, may be sufficient to produce pronounced sinus arrhythmia and sinus bradycardia in an otherwise healthy person.

Report of a Case

One of the authors (P.C.) was the subject of this study. At age 34, he had had 12 years of mountain climbing experience including successful ascents of two peaks higher than 6,000 meters and one summit higher than 7,000 meters. He had experienced the syndrome of acute mountain sickness[3-5] either when ascending in a single day from sea level to about 3,600 meters or on expeditions when first arriving at altitudes of about 5,000 meters.

The present study was undertaken as part of an expedition to Annapurna (8,078 meters) in Nepal. A 24-hour electrocardiographic (ECG) study was done in California on July 20, 1979, at an altitude of 100 meters. Two leads, CC5 and

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CM5, were recorded on an Oxford Medilog 4-24 Holter recorder (Abingdon, England) and scanned on an Oxford Medilog ECG analyzer. Findings included no premature ventricular beats and 2,584 (108 per hour) premature atrial beats. The subject has been known to have frequent premature atrial beats since at least 1966. Heart rate varied from 118 (climbing stairs) to 43 during sleep); longest R-R interval was 1.4 seconds. In sleep, mild sinus bradycardia and sinus arrhythmia were noted. Except for the relatively high frequency of atrial premature beats, these findings are similar to those in a normal population.[6]

On August 12 the subject began the hike to Annapurna base camp. The hike began at 800 meters and on August 24, he reached base camp at 4,350 meters. On August 29 the subject began sleeping at camp I at 5,030 meters. On August 31 a repeat 2 channel Holter study was done for 18.5 hours while sleeping at camp I (5,030 meters) and carrying a load of 17 kg to camp II at 5,560 meters. One premature ventricular beat and 921 (50 per hour) atrial premature beats were noted. At 5,560 meters, sinus rhythm with rates up to 150 was noted while the subject was climbing or shoveling snow.

During sleep, pronounced cyclic sinus arrhythmia was noted. Heart rate was frequently in the 30's with R-R intervals of up to 3.3 seconds (see Figure 1). Rapid changes in heart rate were noted in a cyclic manner.

The subject was receiving no medication at the time of either study.

Later during the expedition, the subject lived for two weeks above 6,090 meters and reached a height of 6,970 meters. The only symptoms of altitude illness were experienced at camp I on September 1, where a severe headache occurred which lasted several hours. This condition resolved following rest and drinking fluids. Further Holter studies were not undertaken because an avalanche occurred, which killed three expedition members and swept away the recording equipment.

Although periodic breathing was not noted in the subject during sleep on Annapurna, it had occurred during sleep at altitudes as low as 3,200 meters on other mountains.

Discussion

Sinus bradycardia in sleep, with sinus arrhythmia and, occasionally, with second degree heart

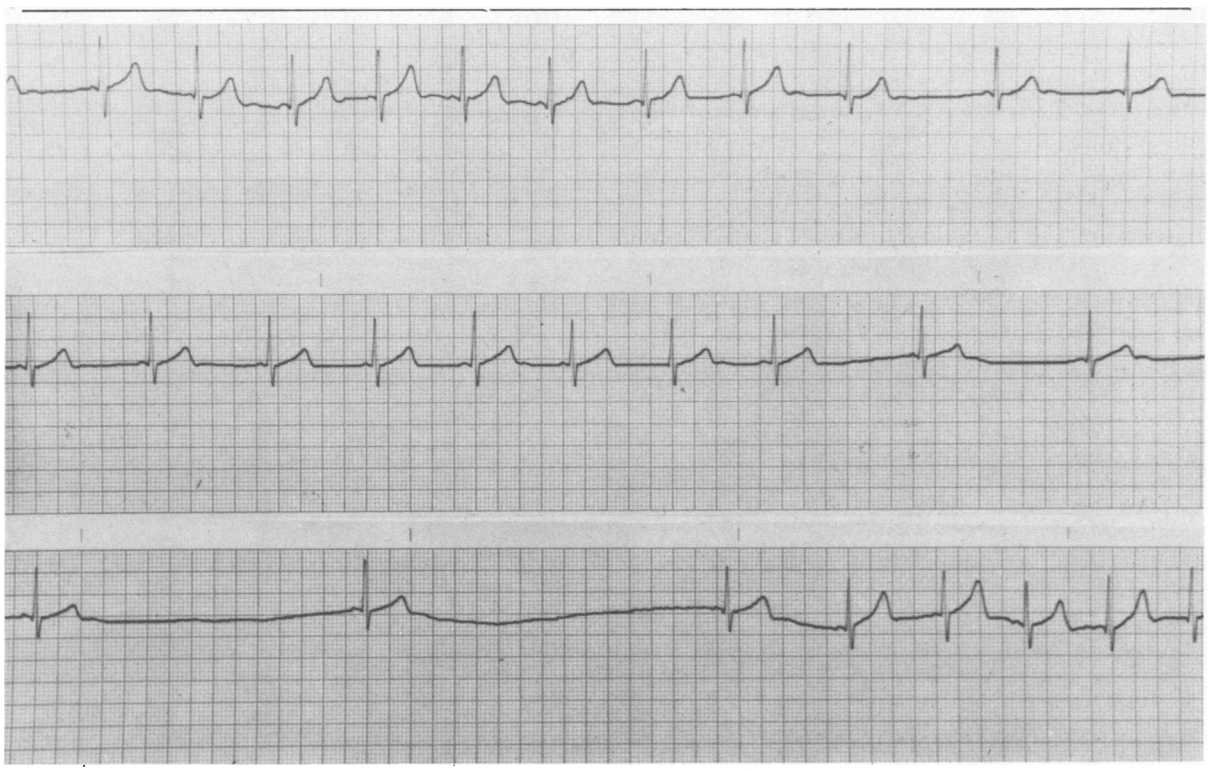

Figure 1.—Continuous electrocardiographic record of lead CM5 recorded in the subject during sleep at an altitude of 5,030 meters. Record shows pronounced sinus arrhythmia with R-R interval up to 3.3 seconds.

block, is a well-known occurrence in normal subjects.[6,7] The reported cases suggest that this is an event only occasionally noted during a sleep study in normal subjects. This has been the experience in our own Holter scanning laboratory. In the present study, however, the pattern is one of pronounced cyclic sinus arrhythmia occurring throughout the night, much like that reported in patients with sleep apnea syndrome. Tilkian and co-workers[1,2] have suggested that this pattern of sleep-associated arrhythmia is virtually diagnostic of the sleep apnea syndrome and that it is almost always associated with upper airway obstruction. They did note, however, that in one of their patients with poliomyelitis the arrhythmic pattern occurred with apnea that was centrally induced and not related to airway obstruction.

In healthy subjects monitored in sleep at 5,360 meters, pronounced periodic breathing has been noted, occurring about 80 percent of the time during sleep. During the apneic periods profound hypoxemia with saturations down to 61 percent were noted. Heart rhythm in these subjects was not reported.[8] In another study at 4,300 meters, periodic breathing was noted during 35 percent of sleep time.[9]

It seems most likely that at 5,030 meters our subject had hypoxia which resulted during sleep in periodic breathing caused by a central nervous system mechanism. During apneic cycles vagal stimulation produced sever sinus bradycardia, while later ventilation and relief of hypoxia resulted in speeding of the sinus rate. This theory is consistent with data recorded by Reite and colleagues[10] at 4,301 meters. They studied sleep at high altitude and noted bradycardia associated with apnea during periodic breathing. The changes they noted were not as dramatic as those in the present study, possibly because the altitude was less. Thus, it appears that hypoxia induced by high altitude can reproduce the arrhythmias seen in the sleep apnea syndrome. Tilkian and co-workers[2] have reported the case of one patient with poliomyelitis who had arrhythmias related to sleep apnea rather than airway obstruction. Even in those patients with upper airway obstruction a central nervous system mechanism plays a role in allowing the obstruction to occur.

Further study of arrhythmia at high altitude is indicated. It is worth noting that in this study it was easy to climb and carry loads with the monitoring equipment. Study of other patient groups with neurologic or cardiopulmonary disorders (or both) and periodic breathing is likely to show similar rhythm disturbances during sleep.

Summary

In a 34-year-old man, pronounced cyclic sinus arrhythmia occurred throughout sleep at 5,030 meters. This arrhythmia is identical to that seen in the sleep apnea syndrome.

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